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Neural Control of Speed Changes in an Opisthobranch Locomotory System

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Abstract. Three forms of forward locomotion have been described in the pteropod mollusk *Clione limacina*, including slow, fast, and escape swimming. The neuromuscular organization of the swimming system suggests that a two-geared system operates for slow and fast swimming, while the escape response is superimposed on fast swimming. In addition to escape, changes in locomotory speed can occur through a dramatic "change-of-gears," or through a more subtle change of speed within gears. The former involves reconfiguration of the central pattern generator and recruitment of previously inactive motor units. The latter can be due to: changes in tonic inputs to the central neurons, central modulation that is not sufficient to "change gears," endogenous properties of muscle cells, and peripheral modulation of muscle contractility. The initial ballistic phase of escape swimming is believed to be triggered by activity in a newly identified pair of swim motor neurons that neither receive information from, nor provide input to, the central pattern generator. These neurons appear to produce a startle response. Evidence presented suggests that most, if not all, of these variables help produce locomotory plasticity in *Clione*.

Introduction

Locomotory speed is a function of several factors, most notably the frequency of movements of locomotory appendages and the force of appendage movements. A change in either of these factors can directly trigger a change in locomotory speed. The former is the provence of the central pattern generator circuitry, whereas the latter can be linked to modifications of the neuromuscular system, and can conceivably include purely peripheral plasticity. Furthermore, activity of central and peripheral modulators can serve to increase the richness of locomotory variability.

Few preparations are conducive to simultaneous electrophysiological monitoring of both central and peripheral activity during both dramatic and subtle changes in propulsive activity. One preparation that combines similar behavioral variability with the typical advantages of the molluscan nervous system—relatively simple neural organization coupled with large cell size—is the locomotory system of the pteropod mollusk *Clione limacina*. Thus far, the majority of work on *Clione* has centered on the central generation of rhythmic locomotory activity (Arshavsky *et al.*, 1985a, b, c, d, 1986, 1989; Satterlie, 1985, 1989; Satterlie and Spencer, 1985; Satterlie *et al.*, 1985), although recent work has focussed on peripheral neuromuscular physiology (Satterlie, 1987, 1988; Satterlie *et al.*, 1990). The purpose of this review is to summarize current work and present new data that relate to the neurobiological basis of locomotory plasticity in the *Clione* swimming system.

Results and Discussion

Locomotory movements of *Clione* include relatively simple two-phase flapping movements of wing-like parapodia (wings). Three forms of locomotion have been described including slow, fast, and escape swimming (Arshavsky *et al.*, 1985a; Satterlie *et al.*, 1985, 1990; Satterlie, 1989). The predominant form is slow swimming, which allows the animal to maintain position in the water column or to move forward (upward) slowly. Wing beat frequencies observed during slow swimming ranged from 1 to 4 Hz. Changes in the rate of forward movement within the slow speed occur both with and without a change in the frequency of wing movements. The latter cases presumably involve changes in wing contractility, as sug-

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gested by behavioral observations in which noticeable changes in the vigor of wing movements have been observed in the absence of a change in wing beat frequency. The change to fast swimming is a triggered, typically dramatic change in the frequency (range: 3–8 Hz) and force of wing movements. In addition to these two basic forms of swimming, a ballistic escape response can be triggered following vigorous stimulation of the tail (Satterlie *et al.*, 1990). The initial phase of escape swimming involves one or two wing cycles characterized by massive contractions of the swim musculature. This "startle" phase is followed by a variable period of enhanced fast swimming. While fast swimming can be triggered without an escape response being activated, escape is always followed by fast swimming.

Despite the three-phase swimming behavior, both the central and peripheral organization of the swimming system appears to be based on two speeds. Evidence presented later suggests that escape swimming is merely superimposed on the fast swimming system. Centrally, the change from slow to fast swimming involves a "change-of-gears," defined here as a change in pattern generator output that results in recruitment (or dropping out) of motor units that have significantly different biochemical and contractile properties than those that were previously (or continuously) active. Peripherally, *Clione* has two types of striated swim muscle fibers: slow-twitch fatigue-resistant and fast-twitch fatigable fibers (Satterlie, 1987; Satterlie *et al.*, 1990). To complement the peripheral organization, two types of swim motor neurons have been described: one associated with slow-twitch muscle activity (and slow swimming), and the other associated with both types of muscle fibers. The latter motor units, which include two large swim motor neurons in each pedal ganglion, are recruited into activity during fast swimming (Satterlie, 1987, 1988, 1989).

With the two-geared arrangement of the *Clione* swimming system before us, three categories of locomotory speed changes will be described, with evidence presented to suggest neurobiological mechanisms for each. Categories of speed change mechanisms include: (1) change-of-gears, (2) change of speed within gears, and (3) escape swimming.

Speed changes due to a "change-of-gears"

Centrally, the change-of-gears from slow to fast swimming involves reconfiguration of the central pattern generator (Arshavsky *et al.*, 1985d, 1989), as previously inactive pedal interneurons become active elements of the swim pattern generator. During slow swimming, a two-phase motor drive is produced by activity in two antagonistic groups of pedal interneurons that interact through reciprocal inhibitory connections (Arshavsky *et al.*, 1985b, c; Satterlie, 1985, 1989). One group of interneurons (V-phase interneurons) produces a single action potential during ventral bending of the wings, whereas the other group (D-phase interneurons) spikes during dorsal bending of the wings. Alternating activity of these two groups of interneurons continues during fast swimming, but two additional interneuron types become active (Arshavsky *et al.*, 1985d, 1989). Delayed V-phase interneurons, which receive only inhibitory input from D-phase interneurons during slow swimming, produce slightly delayed (with respect to normal V-phase interneurons) V-phase spikes during fast swimming. Spikes in the delayed V-phase interneurons trigger activity in the second type of interneuron, called interneurons 12 (Arshavsky *et al.*, 1985d, 1989). Each interneuron 12 produces a plateau potential that is turned on by excitatory input from delayed V-phase interneurons, and is turned off by inhibitory input from D-phase interneurons. Plateau potentials of interneurons 12 inhibit V-phase interneurons and excite D-phase interneurons. Addition of the delayed V-phase and type 12 interneurons to the swim pattern generator thus produces an early termination of V-phase activity coupled with onset of the next D-phase. This change increases the cycle frequency of pattern generator output (Arshavsky *et al.*, 1985a) and is associated with a recruitment of previously inactive large motor neurons (Fig. 1). As mentioned previously, recruitment of these motor neurons is associated with the activation of the fast-twitch musculature of the wings. The change-of-gears is also associated with a 5–15 mV tonic depolarization in "normal" D- and V-phase interneurons of the swim pattern generator (Fig. 1). The combination of increased cycle frequency and increased force of wing contractions through recruitment of "fast-twitch" motor units produces a dramatic increase in forward propulsion speed.

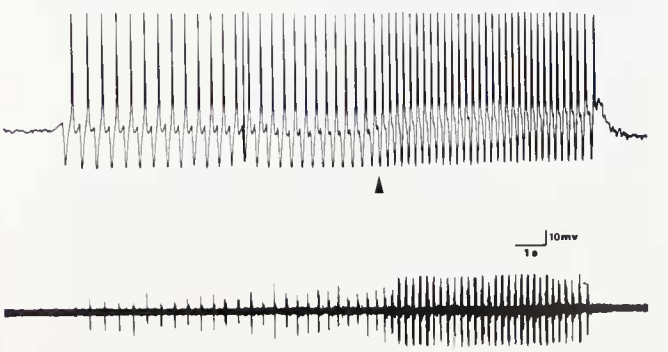

Figure 1. Intracellular recording from a pattern generator interneuron of *Clione* (top trace) with a simultaneous extracellular recording from the wing nerve (bottom trace). The record shows a change-of-gears (arrow) involving an increase in cycle frequency and a tonic depolarization in the interneuron. The change recruits large spikes in the wing nerve recording. These large spikes have been shown to reflect activity in large swim motor neurons. Recording by A. N. Spencer, University of Alberta.

Rewiring of a central pattern generator is certainly not a new concept. The pyloric central pattern generator of the lobster stomatogastric system can exhibit at least four distinct functional circuits and thus four distinct motor activities (Flamm and Harris-Warrick, 1986a, b; Harris-Warrick *et al.*, 1989). In addition to the unmodulated circuit, the amine modulators dopamine, octopamine, and serotonin can each produce a dramatically distinct functional circuit (see Harris-Warrick *et al.*, 1989, for a review). In the opisthobranch mollusk *Tritonia*, variable output in the body wall motor systems can be produced by varying the types and intensities of triggering sensory inputs. According to the polymorphic network concept (Getting and Dekin, 1985), different inputs can activate different configurations of motor control systems to produce unique motor outputs as distinctive as body wall withdrawal and swimming movements. These two examples demonstrate that a motor control system, or part of it, can be used for more than one behavior. In comparison, reconfiguration of the *Clione* swim pattern generator appears to involve exclusively frequency modulation rather than changes in the phase relationships or functional wiring of the pattern generator.

The change from slow to fast swimming in *Clione* is induced in both intact and reduced preparations when the preparations are bathed in 10^{-5} to 10^{-6} *M* serotonin (Arshavsky *et al.*, 1985a, d; Satterlie 1989). Under these conditions, fast swimming continues as long as serotonin remains in the bath. At the level of individual pattern generator interneurons, serotonin produces a 5–10 mV tonic depolarization similar to that seen during spontaneous fast swimming. The source of these tonic depolarizations is not known. Serotonin has also been implicated in the initiation of swimming activity in the leech (Kristan and Weeks, 1983; Nusbaum and Kristan 1986; Nusbaum, 1986) and of *Aplysia brasiliana* (Parsons and Pinsker, 1989), as well as pedal locomotion in non-swimming *Aplysia* (Mackey and Carew, 1983). Serotonin also modulates ongoing rhythmic activity in a number of preparations, including lamprey swimming (Harris-Warrick and Cohen, 1985), feeding in *Aplysia* (Kupfermann and Weiss, 1982), insect flight (Claassen and Kammer, 1986), and the pyloric rhythm of the lobster (Flamm and Harris-Warrick, 1986a, b). Serotonin can also have system-wide behavioral effects, as in the regulation of posture in lobsters (Kravitz *et al.*, 1985).

Changes of swimming speed within gears

Although the possibilities for changes of speed within gears are numerous, four possibilities will be considered here: (1) changes in tonic input to swim interneurons and motor neurons, (2) central modulation of the pattern generator (*e.g.*, with serotonergic inputs) at a level not sufficient to change gears, (3) the role of endogenous properties of muscle cells, and (4) peripheral modulation of muscle contractility. The first two involve central modifications while the last two modify peripheral activity.

Changes in tonic input to swim neurons

Despite the description of pedal neurons that show variable tonic activity associated with changes in pattern generator activity in *Clione* (Arshavsky *et al.*, 1984), little is known about the variety and sources of tonic influences over pattern generator activity. Inasmuch as tonic depolarization of isolated pattern generator interneurons is related to spontaneous firing frequency (Arshavsky *et al.*, 1986), then tonic inputs can presumably modify the frequency of pattern generator output. Provided that the inputs do not cause pattern generator reconfiguration, the change in cycle frequency will be translated into a change of locomotory speed within the appropriate "gear." Tonic input could exert this influence in either slow or fast swimming gears.

Central modulation not sufficient to change gears

The source of central serotonergic inputs to the pattern generator that are responsible for reconfiguration and gear change have not yet been identified. But circumstantial evidence now in hand has led us to investigate descending serotonergic inputs from the cerebral ganglia. Serotonin-immunoreactive neurons have been found in the medial posterior and medial anterior regions of the cerebral ganglia. Axons from some of these cells run from the cerebral ganglia to the pedal ganglia via the cerebro-pedal connectives. Focal extracellular stimulation of the medial posterior region of a pedal ganglion results in acceleration of pattern generator activity, or with strong stimuli, changes in pattern generator activity identical to changes associated with activation of fast swimming activity. Transection of the cerebro-pedal connective greatly reduces these responses. Assuming that the central modulation does not operate in an all-or-none manner, subthreshold levels of modulation (subthreshold for change of gears) might trigger a change of swimming speed within the slow gear, and different levels of supra-threshold modulation might produce variable pattern generator activity in the fast gear. Such changes of swimming speed should be expressed as a change in cycle frequency, unless swim motor neurons are also affected by the central modulatory subsystem. In the latter case, changes in both cycle frequency and force of wing movements will be seen. A further, purely speculative possibility allows for separate modulation of pattern generator interneurons and swim motor neurons, a condition that would add greatly to the complexity of the behavioral output. Potential central

modulators other than serotonin are not being considered here, but should not be discounted.

Intrinsic properties of muscle cells

Intrinsic properties of muscle cells, particularly related to repetitive firing activity, can influence the force of swim muscle contractions. Such intrinsic properties could be synaptic or non-synaptic, the latter including changes in passive or active membrane properties, or in excitation-contraction coupling. Both slow-twitch and fast-twitch fibers of the *Clione* swimming system exhibited non-synaptic facilitation of the amplitude of spike-like responses with repetitive, direct depolarization of individual muscle cells (Satterlie, 1988). The facilitation was strongly frequency-dependent, so that both overall amplitude of spike-like responses and initial rate of change of spike-like response amplitude showed a positive correlation with frequency of induced activity over the range of frequencies normally encountered during slow and fast swimming (in prep.). Provided that the contractile force of whole muscles is related to changes in spike-like response amplitude recorded from individual cells, overall muscle force should change in parallel with changes in pattern generator frequency.

Peripheral modulation of contractile force

A cluster of 7–10 serotonin-immunoreactive neurons have been found in the medial margin of each pedal ganglion of *Clione* (Fig. 2). At least two neurons from this cluster send axons to the ipsilateral wing via the wing nerve. Induced activity in these two neurons produced no direct motor response; but when activity was triggered during ongoing swimming activity, muscle contractions

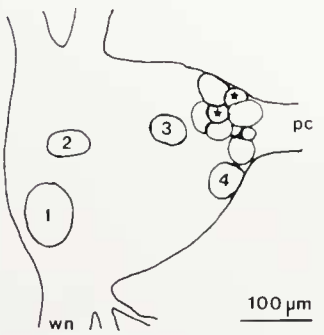

Figure 2. Schematic diagram of the dorsal surface of the left pedal ganglion of *Clione*. The two large motor neurons (major landmarks of the ganglion) are indicated by cells 1 and 2. Cells 3 and 4 represent motor neurons that initiate escape swimming. The remaining cells represent serotonin-immunoreactive cells. The two cells marked with an asterisk have been electrophysiologically identified; they send axons into the ipsilateral wing via the wing nerve (wn). These cells enhance muscle contractility as shown in Figure 3. pc–pedal-pedal commissure.

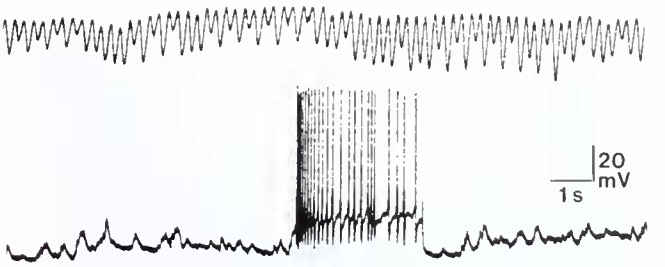

Figure 3. Dual recording from a serotonin-immunoreactive neuron (bottom trace) and a wing force transducer (top trace—not calibrated). Following a burst of action potentials in the neuron, muscle contractions are enhanced. The latency of the response is approximately one second, and the duration is 5 s. The neuron was hyperpolarized by a −1 nA current during the recording to prevent spiking. The burst was triggered by switching to a +1 nA current.

were enhanced (Fig. 3). The response latency was approximately one second from the initiation of the induced burst, and the effect lasted from 3–10 s. Preliminary evidence suggests that this enhancement was due to an increased amplitude of the spike-like response in some, but not all, of the muscle cells.

Peripheral modulation, including both pre- and post-synaptic effects, have been noted in numerous preparations (*e.g.*, Kravitz *et al.*, 1985; Kobayashi and Hasimoto, 1982; Maranto and Calabrese, 1984; Weiss *et al.*, 1978). Induced bursts in the pedal serotonin-immunoreactive neurons of *Clione* produced no apparent synaptic activity in either pattern generator or motor neurons, and produced no changes in frequency or intensity of spike activity in either neuron type. This suggests an interesting dichotomy in serotonin modulation of swimming in *Clione*: *i.e.*, pedal serotonin-immunoreactive neurons modulate muscle activity, whereas proposed cerebral serotonergic neurons modulate pattern generator activity. A similar separation of central and peripheral modulation is seen in the leech heartbeat system (Calabrese and Arbas,

Table I

Summary of four possible modulatory states in the swimming system of Clione limacina *based on separate central and peripheral modulatory subsystems*

Modulatory state	Swimming activity
No modulation	Slow swimming, normal muscle contractility
Peripheral modulation only	Slow swimming, enhanced muscle contractility
Central modulation only	Fast swimming, normal muscle contractility
Central and peripheral modulation	Fast swimming, enhanced muscle contractility

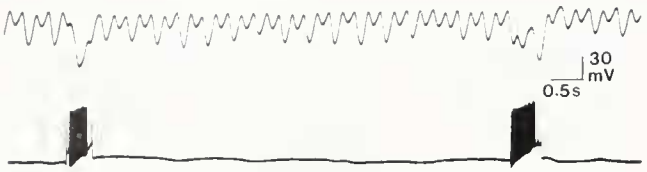

Figure 4. Dual recording from a "startle" motor neuron (bottom trace) and an uncalibrated wing force transducer (top trace). Note the absence of pattern generator input to the neuron despite the ongoing swimming activity. Bursts of action potentials were triggered in the neuron with +12 nA injected currents (through the recording electrode). The resultant bursts of activity induced strong contractions of the wing.

1985). Assuming the simplest case of supra-threshold modulation in both central and peripheral subsystems of *Clione*, the separation of pattern generator and muscle modulatory subsystems allows four possible states with respect to serotonin modulation of swimming activity (Table 1). As mentioned previously, central modulation will primarily affect cycle frequency, whereas peripheral modulation will affect contractile force.

This discussion takes into account only one peripheral modulatory system. The possibility of other modulatory inputs, as well as the release of multiple transmitters or modulators from the serotonin-immunoreactive neurons, could add further complexity to the swimming system.

Escape swimming

An interesting pair of motor neurons have recently been identified from each pedal ganglion of *Clione* (Fig. 4). These motor neurons activate both slow-twitch and fast-twitch fibers of the swim musculature, but do not receive input from the swim pattern generator. The neurons were originally overlooked, because they are electrically silent during normal swimming activity and have extremely high firing thresholds. In some preparations, it is very difficult to stimulate electrical activity from these cells with intracellular current injection. Induced bursts of spikes in the motor neurons produce massive contractions of the ipsilateral wing. Despite this strong peripheral input, the cells have no inputs to, or influence over, the activity of interneurons of the pattern generator or swim motor neurons. Their powerful effect on swim musculature, their total independence from the swim pattern generator, and their high firing threshold suggest that these motor neurons may participate in the primary phase of escape swimming by triggering the initial ballistic movement; indeed, the ballistic movement may function as a startle response. The maintenance of escape swimming, involving the variable period of enhanced fast swimming, could represent activation of both central and peripheral serotonergic modulatory subsystems (see Table 1). Multiple recordings from "startle" neurons and serotonin-immunoreactive neurons following tail stimulation in intact preparations have not yet been completed due to technical difficulties, but should help clarify this relationship.

The foregoing discussion introduces several levels at which changes of locomotory speed can occur in the swimming system of *Clione*. Some of the results are preliminary, while a few are purely speculative. It is clear, however, that both central and peripheral modulatory influences are operating, and that significant changes in both frequency and strength of wing movements can contribute to locomotory speed changes. With this information, we are beginning to gain an appreciation for the neurobiological complexity involved in locomotory plasticity in this relatively "simple" swimming system. Our comprehension of the neuronal bases of speed changes involves changes of gears, changes of speed within gears, and superimposed inputs, such as escape. This understanding is providing a good starting point for further investigation of other forms of input and modulation, as well as detailed descriptions of the intrinsic properties of all cells involved in swimming behavior.

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